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CORRESPONDENCE

Erosive pustular dermatosis of the scalp in an elderly patient

Dear Editor,

An 89-year-old man presented with pustules superimposed on painful erythematous plaques covering his scalp after a recent minor abrasion. Despite aggressive debridement, changing dressing, and administration of systemic antibiotics for 4 weeks, the lesions progressed extensively. A physical examination revealed numerous small pustules studded on the yellowish-crusted plaques and superficial erosions. Detachment of the crusts exhibited erosive patches with atrophic skin change (Figure 1). Repeated cultures from the pustules were unable to demonstrate bacterial or mycological microorganisms. Laboratory investigations showed leukocytosis (15,500/mcL) and elevated levels of C-reactive protein (12 mg/dL). Both serum rapid plasma reagin test and human immunodeficiency virus screening test results were nonreactive. Rheumatoid factor and antineutrophil cytoplasmic antibody were also negative. The titer of antinuclear antibody was slightly elevated (1:40) with a mixed staining pattern.

A skin biopsy of the pustular lesions disclosed a dense neutrophilic infiltration and subcorneal microabscesses. In the papillary dermis, diffuse leukocytoclasia and severe papillary edema were observed (Figure 2A). Vasculitis was not apparent. The inflammatory infiltrate spread into the deep dermis, and the follicles and sebaceous glands displayed remarkable necrosis with infiltration of neutrophils, mononuclear cells, and plasma cells (Figure 2B). Periodic acid–Schiff stains and direct immunofluorescence study results were negative. Based on the clinical and histological presentations, a diagnosis of erosive pustular dermatosis (EPD) of the scalp was made. We then initiated treatments including oral prednisolone (10 mg/d), topical fusidic acid, and desoximetasone 0.25% ointment. The lesions recovered and only a small area of erosion remained in the 2-month follow-up.

In 1979, Pye et al[1] reported six patients with previously undescribed inflammatory dermatosis confined to the scalp, and characterized by sterile pustules, erosions, crusts, and macerated keratin formation, which they termed “erosive pustular dermatosis.” EPD is characteristically reported in elderly female patients, beginning as localized amicrobial pustular lesions, which over a period of months to years can evolve into large eroded areas covered by superficial crusts. Scarring alopecia may occur after a slow and protracted course. The pathogenesis of EPD remains uncertain, but in most cases, a history of local trauma has been documented.[2] Other predisposing factors have included local treatment (cryotherapy, radiotherapy, photodynamic therapy, laser therapy, topical retinoic acid, fluorouracil, or imiquimod), previous varicella zoster viral infection, sun-burn injuries, chronic nonhealing burn wounds, the administration of epidermal growth factor receptor antagonists (e.g., gefitinib), and other associated autoimmune diseases.[3–6]

The histological features of EPD vary according to the biopsy site. The epidermis may show hyperkeratosis, atrophy, and occasionally subcorneal pustules formation. The dermis may display chronic inflammation with lymphoplasma cells and neutrophils. Destruction of hair follicles and pilosebaceous units can also occur. In the later stages, foreign-body giant cells and dermal fibrosis are observed. Direct immunofluorescence tests are usually negative. The clinical differential diagnosis of pustulosis of the scalp is diverse, including bacterial or fungal infections, folliculitis decalvans, dissecting cellulitis, pustular psoriasis, subcorneal pustular dermatosis, pyoderma gangrenosum, pemphigus, cicatricial pemphigoid, discoid lupus erythematosus, lichen planopilaris, superficial granulomatous pyoderma, and field cancerization.[3–5] To establish the diagnosis of EPD of the scalp, clinicopathological correlations and careful exclusion of other diseases are essential. EPD can also occur on the legs of elderly patients with chronic venous insufficiency or edematous skin changes.[5] Leg lesions are similar to those on the scalp with erythematous moist erosions and pustules. The infectious process should be carefully evaluated in these clinical situations.

Figure 1 Extensive crusted and erosive areas with numerous pustular lesions.

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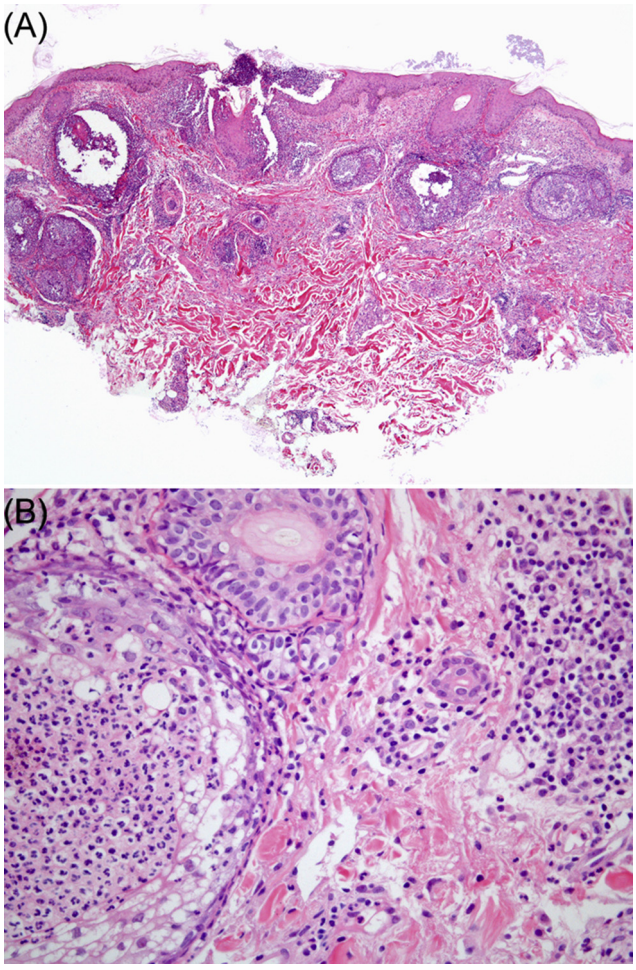

Figure 2 (A) Diffuse neutrophilic infiltration leading to subcorneal microabscess formation, papillary edema, and necrosis of hair follicles and sebaceous glands (hematoxylin and eosin; original magnification, 40×). (B) High-power view showing inflammation of the pilosebaceous units with numerous neutrophils, mononuclear cells, and plasma cells (hematoxylin and eosin; original magnification, 400×).

Therapeutic approaches to EPD include the topical administration of corticosteroids, calcipotriol, tacrolimus, and dapsone gel[4,5] or oral administration of corticosteroids, isotretinoin, dapsone, and zinc sulfate.[3–5] Cryotherapy and photodynamic therapy have also been applied.[3,5] However, EPD is a chronic and relapsing condition that requires long-term management. Prolonged topical application of potent steroids should be used with caution, especially for the atrophic skin. In addition, relapse of EPD after discontinuation of topical steroids has been reported.[1] Thus, to reduce relapse and to avoid further skin atrophy, topical calcineurin inhibitors are considered as a maintenance therapy.[5]

Herein, we reported the case of an elderly patient who developed typical presentations of EPD of the scalp after minor abrasion trauma. In summary, EPD is an uncommon diagnosis, which classically affects elders, and causes sterile pustulosis, crusts, and eroded atrophic skin on the scalp or legs. Predisposing factors and events usually exist. Histopathology may show a variable degree of lymphoplasma and neutrophil cell infiltration, and follicular or pilosebaceous destruction. Chronic relapsing inflammation and delayed wound healing are characteristic features of EPD, and long-term follow-up is indicated.

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